

HOSPITAL CHRONICLES 2006, 1(1): 9–19

REVIEW

The Spectrum of Acquired Atrioventricular Block in Clinical Practice

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KEY WORDS: *Type I second-degree atrioventricular block, Type II second-degree atrioventricular block, Atrioventricular Block, Electrocardiography, Cardiac pacemaker, Cardiac pacing*

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ABSTRACT

Type I and type II second-degree AV block characterize block of a single sinus P wave: Type I block describes visible, varying and generally decremental AV conduction and type II block describes apparent all-or-none conduction without visible changes in AV conduction time before and after the blocked impulse. Absence of sinus slowing is an important criterion of type II block because a vagal surge (generally benign) can superficially resemble type II block. The diagnosis of type II block cannot be established if the first postblock P wave is followed by a shortened PR interval or is not discernible. All correctly defined type II blocks are infranodal. A pattern resembling narrow QRS Type II block together with an obvious type I structure in the same recording effectively rules out type II block because the co-existence of both types of narrow-QRS block is rare. Narrow QRS type I block is almost always AV nodal whereas type I block with bundle branch block outside acute myocardial infarction is infranodal in 60-70 % of cases. 2:1 AV block cannot be classified in terms of type I or II blocks and can be nodal or infranodal.

Pacing is indicated in symptomatic marked first-degree AV block (>0.30 sec.), but patients with systolic heart failure might benefit more with biventricular pacing.

Permanent pacing is almost never needed after inferior myocardial infarction and narrow QRS AV block. It should be considered only if second- or third-degree AV block persist for 14-16 days. Patients with bundle branch block and transient second- and third-degree AV block during anterior myocardial infarction have a high risk of sudden death after hospital discharge usually from ventricular tachyarrhythmias rather than AV block. They should receive an implantable cardioverter- defibrillator rather than a stand-alone pacemaker in the setting of severely depressed systolic left ventricular function.

There are many causes of atrioventricular (AV) block but progressive idiopathic fibrosis of the conduction system related to an aging process of the cardiac skeleton is the most common cause of chronic acquired AV block. Barring congenital AV block, Lyme disease is the commonest cause of reversible third-degree AV block in young individuals and it is usually AV nodal. Before implantation of a permanent pacemaker, reversible causes of AV block such as Lyme disease, hypervagotonia, athletic heart, sleep apnea, ischemia, and drug, metabolic, or electrolytic imbalance must be excluded. Table 1 outlines the format used in the 2002 American College of

TABLE 1. Standard ACC/AHA/NASPE Format for Device Indications.

Class I:	Conditions for which there is evidence and/or general agreement that a given procedure or treatment is beneficial, useful, and effective.
Class II:	Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.
Class IIa:	Weight of evidence/opinion is in favor of usefulness/efficacy.
Class IIb:	Usefulness/efficacy is less well established by evidence/opinion.
Class III:	Conditions for which there is evidence and/or general agreement that a procedure/treatment is not useful/effective, and in some cases may be harmful.

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Cardiology/American Heart Association/ North American Society of Pacing and Electrophysiology (ACC/AHA/NASPE) guidelines for pacemaker implantation [1]. The indications for permanent pacing in second- or third- degree AV block unlikely to regress are often straightforward in symptomatic patients but they are more difficult in asymptomatic patients. Some of the ACC/AHA/NASPE guidelines appear somewhat dogmatic. The final responsibility rests with the physician in terms of decisions, and the guidelines only represent a basic framework to start from.

ANATOMIC AND ELECTROPHYSIOLOGIC CONSIDERATIONS

The specialized conduction system below the AV node consists of the bundle of His proximally and the 3 intraventricular fascicles distally — The right bundle branch, the anterior (superior) division of the left bundle branch, and the posterior (inferior) division of the left bundle branch (Fig. 1). Although the left bundle branch does not exist as two discrete anatomic divisions, the functional separation into two fascicles is a useful concept clinically and electrocardiographically. Left anterior hemiblock (LAH) causes left axis deviation (-45 degrees or more superior) or an axis in the right superior quadrant, and left posterior hemiblock (LPH) causes right inferior axis deviation in the frontal plane. Disease in any one of the 3 fascicles is of little clinical significance. When the ECG shows evidence of block in 2 fascicles (bifascicular block), only one pathway remains for AV conduction but AV block does not occur. Bifascicular block can be identified on the ECG when there is right bundle branch block (RBBB) with LAH, RBBB with LPH or left bundle branch block (LBBB) regardless of the axis in the frontal plane. In LBBB, the frontal

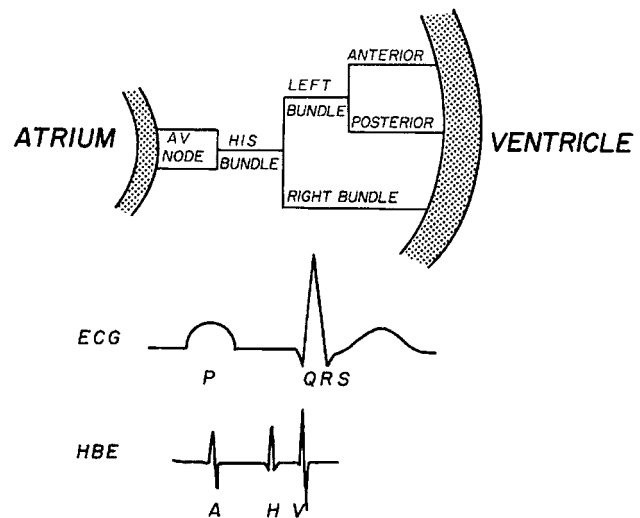


FIGURE 1. Diagrammatic representation of the atrioventricular conduction system. The bundle branch system consists of a three pronged system (2 on the left side and 1 on the right side). At the bottom, the surface ECG is depicted simultaneously with a His bundle electrogram (HBE). The HBE is recorded near the septal leaflet of the tricuspid valve. A – reflects depolarization of the low atrium, H – depolarization of the His bundle, and V – depolarization of the upper ventricular septum. The AH interval reflects AV nodal conduction and the HV interval reflects conduction within the His-Purkinje system. The normal HV interval is 35-55 msec. A prolonged HV interval indicates delayed conduction in all three prongs of the conduction system and/or the His bundle. With His-Purkinje disease the HV interval (but not the surface QRS complex) remains normal as long as one of the three prongs conducts normally. (Barold SS. Pacemaker treatment of bradycardias, and selection of optimal pacing modes. In: Zipes DP (Ed.). Contemporary Treatments in Cardiovascular Disease, 1997; 1:123. with permission).

plane axis carries little value for the diagnosis of hemiblock. Right axis deviation with LBBB is rare and usually indicates the presence of a severe end-stage dilated cardiomyopathy while patients with LBBB and left axis deviation generally have more advanced myocardial disease than those with LBBB and a normal axis. In bifascicular block without documented second- or third- degree AV block, the functional status of the third fascicle cannot be evaluated from the ECG. During 1:1 AV conduction the diagnosis of trifascicular block can only be made in rare situations involving alternating RBBB and LBBB or RBBB with alternating LAH and LPH [2].

INTRACARDIAC RECORDINGS

The His bundle potential is easily recorded with an electrode catheter passed from the femoral vein to the level

of the tricuspid valve. The “A” wave reflects depolarization of the low right atrium, the “V” deflection is produced by ventricular depolarization near the recording catheter and the “H” deflection is generated by rapid transmission of depolarization through the bundle of His and appears between the A and V waves (Fig. 1). The AH interval (normal is 60 to 140 ms) basically reflects AV nodal conduction (rarely intra-atrial delay) and the HV interval (normal is 35-55 ms) reflects the conduction time from the His bundle to the beginning of ventricular activation—that is the His-Purkinje system. Bifascicular block is associated with a normal HV interval if conduction in the third fascicle remains intact. Consequently a prolonged HV interval strongly suggests delayed conduction in all 3 fascicles. However HV delay may occasionally be within the His bundle itself or due to a combination of delay in the His bundle and the 3 fascicles. Prolongation of the PR interval in the ECG is an unreliable indicator of bifascicular or trifascicular disease because it may be due to prolongation of the AH and/or HV intervals. Consequently RBBB + LAH + 1st degree AV block should not be designated as trifascicular block unless invasive recordings demonstrate prolongation of the HV interval. Electrocardiographic documentation of true trifascicular block during 1:1 AV conduction is rare and involves either alternating or bilateral bundle branch block or fixed RBBB with alternating LAH and LPH. Finally asymptomatic patients with bundle branch block or bifascicular block do not require determination of the HV interval because the risk of developing complete AV block is very low at about 1-2 % per year.

COMPLETE AV BLOCK

The 1998 ACC/AHA guidelines designate asymptomatic complete AV block with ventricular escape rates >40 bpm as a class II indication for pacing [1]. The rate criterion of >40 bpm is arbitrary and unnecessary. It is not the escape rate that is critical to stability, but rather the site of origin of the escape rhythm [junctional or ventricular]. Rate instability may not be predictable or obvious. Irreversible acquired complete AV block should be a class I indication for pacing. In neuromuscular disease such as myotonic dystrophy, pacing should be considered much earlier in the course of the disease and offered to the asymptomatic patient once any conduction abnormality is noted and subsequent follow-up shows progression even when second-degree AV block has not yet developed. Waiting for the development of complete AV block may expose patients to a significant risk of syncope or even sudden death.

SECOND-DEGREE AV BLOCK

Type I block and type II second-degree AV block are

electrocardiographic patterns and as such should not be automatically equated with the anatomic site of block.

TYPE I BLOCK (WENCKEBACH OR MOBITZ TYPE I)

Type I second-degree AV block is defined as the occurrence of a single nonconducted sinus P wave associated with inconstant PR intervals before and after the blocked impulse provided there are at least two consecutive conducted P waves (i.e., 3:2 AV block) to determine behavior of the PR interval [3]. The PR interval after the blocked impulse is always shorter if conducted to the ventricle (Fig. 2). The term “inconstant” PR or AV intervals is important because the majority of type I sequences are atypical and do not conform to the traditional teaching about the mathematical behavior of the PR intervals [4-6]. The description of “progressive” prolongation of the PR interval is misleading because PR intervals may shorten or stabilize and show no discernible or measurable change anywhere in a type I sequence (Fig. 2). Indeed, atypical type I structures in their terminal portion can exhibit a number of consecutive PR intervals showing no discernible change

TABLE 2. Pitfalls in diagnosis and treatment of AV block.

- Type I AV block can be physiological in athletes resulting from heavy physical training
 - Type I AV block can be physiological during sleep in individuals with high vagal tone
 - Failure to suspect vagally-induced AV block, i.e. vomiting
 - Failure to recognize reversible causes of AV block
 1. Lyme disease
 2. Electrolyte abnormalities
 3. Inferior myocardial infarction
 4. Sleep apnea
 - Poor correlation between narrow QRS type I block and symptoms.
 - Beliefs that all type I blocks with a wide QRS complex are AV nodal.
 - Nonconducted atrial premature beats masquerading as AV Block.
 - What appears to be narrow QRS type II block may be a type I variant.
 - Atypical type I sequence mistaken for type II block.
 - Making the diagnosis of type II block without seeing a truly conducted postblock P wave (shortage of PR intervals).
 - A recording that appears to show both types I and II and a narrow QRS complex may in fact represent only type I block.
 - Concealed extrasystoles causing pseudo-AV block. (Look for associated unexpected sudden PR prolongation, combination of what appears to be type I and type II and isolated retrograde P waves from retrograde conduction of the concealed extrasystole).
 - Relying solely on a computer-rendered ECG diagnosis: Computer interpretations are notoriously error-prone.
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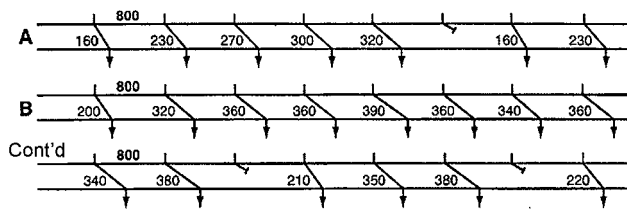


FIGURE 2. Diagrammatic representation of AV block. The 3 levels represent activation of the atria, AV junction (PR intervals are shown between the lines), and ventricles respectively. All values are in ms. (A) Classic type I AV block. (B) Relatively long and atypical type I sequence. Note the irregular fluctuations of the PR intervals before the dropped beat. Reproduced with permission from reference 11.

before the single blocked beat [4]. In such an arrangement the post-block PR interval is always shorter (Fig. 3A, 4A). Slowing or an increase of the sinus rate does not interfere with the diagnosis of type I block.

Increments in AV conduction

Increments in AV conduction (AH interval) in Type I AV nodal block are typically large. Type I infranodal block typically exhibits small increments in AV conduction (HV interval) and large increments in AV conduction occur uncommonly, but they may occasionally be so tiny that they superficially mimic type II second-degree AV block. In contrast type I infranodal block (discussed below) typically exhibits small increments in AV conduction (HV interval) and large increments in AV conduction occur uncommonly.

Site of Block: In narrow QRS type I block, the block is in

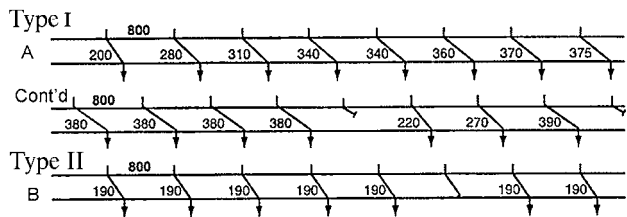


FIGURE 3. Diagrammatic representation of various forms of second-degree AV block with the same format as in Fig 2. (A): Relatively long and atypical type I sequence with several constant PR intervals before a dropped beat. Note the shorter PR interval after the blocked P wave. This pattern should not be called type II AV block. It is essential to examine all the PR intervals in long rhythm strips and not merely several PR intervals preceding a blocked impulse. (B): True type II AV block. Every atrial impulse successfully traverses the AV node which is not afforded a long recovery time as occurs in type I AV block. Note that the PR interval after the blocked beat is unchanged. Reproduced with permission from reference 11.

the AV node in almost all the cases. Type I block can be physiological especially during sleep in normal individuals with high vagal tone and these people need no treatment. Asymptomatic type I second-degree AV block present throughout the day is generally considered benign. However, some workers in Britain recommend permanent pacing in this setting for prognostic reasons based on long-term mortality data from a single center [7-9]. We believe that these observations need to be confirmed before recommending pacing for this situation.

IntraHisian narrow QRS type I block is rare. In practice, cases of narrow QRS intraHisian type I block due to chronic conduction system disease are not usually found because virtually all narrow QRS type I blocks are dismissed as being AV nodal. IntraHisian block although rare clinically, may be provoked by exercise in contrast to type I AV nodal block which generally improves with exercise. Improvement of AV block with exercise is highly suggestive of AV nodal second-degree AV block. His bundle recordings are unnecessary in an asymptomatic patient with narrow QRS type I block. However if an electrophysiologic study (performed for other reasons) in

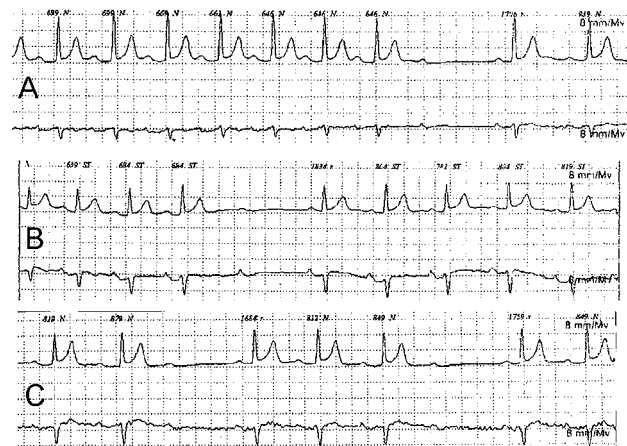


FIGURE 4. Representative Holter recordings from an asymptomatic patient with a type I block variant that was misdiagnosed as type II block by several physicians. A: Type I block with constant PR intervals before the blocked beat. Note that there is a slight increase in the sinus rate in the sequence before the blocked beat. However, the sinus rate then slows and the blocked P wave occurs in association with sinus slowing a combination consistent with a vagal phenomenon. The PR intervals after the blocked beat are inconstant. B: Type I variant simulating type II block. The PR intervals are constant before and after the blocked beat. However, there is obvious sinus slowing simultaneously with the nonconducted P wave. C: Type I block. Note that in the presence of a narrow QRS complex, the occurrence of type I (with fairly large increments of the PR intervals) and what appears to be type II block basically rules out the presence of a true type II block.

such a patient reveals infranodal block, a pacemaker should be recommended as a class I indication because diffuse His-Purkinje disease is likely to be present.

Type I second-degree AV block with bundle branch block (which is far less common than narrow QRS type I block) must not be automatically labeled as AV nodal. Outside of acute myocardial infarction, type I block and bundle branch block ($QRS \geq 0.12$ s) occurs in the His-Purkinje system in 60-70% of the cases [10] (Fig. 5). In such cases exercise is likely to aggravate the degree of AV block. Yet, many still believe that type I blocks are all AV nodal and therefore basically benign. It is believed that the prognosis of infranodal type I block is as serious as that of type II block and a permanent pacemaker is generally recommended in both types regardless of symptoms. On this basis, patients with type I block and bundle branch block should undergo an invasive study to determine the level of second-degree block in the conduction system.

However, it is unknown whether underlying right bundle branch block (unifascicular block) is prognostically different from underlying left bundle branch block (bifascicular block) in the setting of asymptomatic type I second-degree infranodal block.

TYPE II BLOCK (MOBITZ TYPE II)

The definition of type II second-degree AV block continues to be problematic in clinical practice [3,11,12]. Type II second-degree AV block is defined as the occurrence of a single non-conducted sinus P wave associated with constant

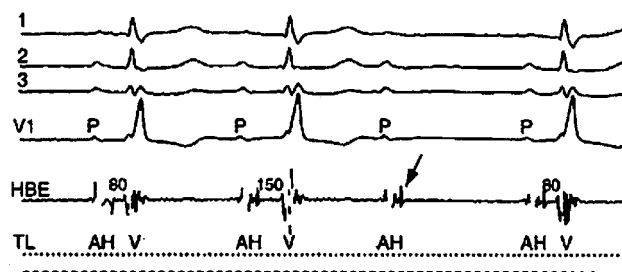


FIGURE 5. Sinus rhythm with second-degree Type I 3:2 infranodal AV block, and right bundle branch block. Note that the AH interval remains constant. The HV interval increases from 80 ms (following first P wave) to 150 ms (following second P wave). The third P wave is followed by an H deflection but no QRS complex. AV block occurs in the His-Purkinje system below the site of recording of the His bundle potential (arrow). Note the shorter PR interval after the nonconducted P wave, a feature typical of Type I second-degree AV block. HBE = His bundle electrogram, A = atrial deflection, H = His bundle deflection, V = ventricular deflection, P = P wave. TL = time lines 50 ms. (Barold SS. Pacemaker treatment of bradycardias and selection of optimal pacing modes. In: Zipes DP (Ed.). Contemporary Treatments in Cardiovascular Disease, 1997; 1:123, with permission).

PR intervals before and after the blocked impulse, provided the sinus rate or the P-P interval is constant and there are at least two consecutive conducted P waves (i.e., 3:2 AV block) to determine behavior of the PR interval [13,14]. The pause encompassing the blocked P wave should equal two (P-P) cycles (Fig. 3B, 6). The PR interval is either normal or prolonged but remains constant. Type II block cannot be diagnosed whenever a single blocked impulse is followed by a shortened post-block PR interval or no P wave at all. In this situation it is either a type I pattern or an unclassifiable sequence. Stability of the sinus rate is a very important criterion because a vagal surge can cause simultaneous sinus slowing and AV nodal block, generally a benign condition that can superficially resemble Type II second-degree AV block [3]. In the presence of sinus arrhythmia, the diagnosis of type II block may not be possible if there is sinus slowing especially if the block occurs in one of the longer cycles. In contrast the diagnosis of type II block is possible with an increasing sinus rate.

The 2002 ACC/AHA/NASPE guidelines introduced a new classification of type II second-degree AV block: wide QRS type II block (which makes up 65-80 % of type II blocks) with a class I indication for pacing and narrow QRS type II block with a class II indication for permanent pacing [1]. This differentiation is strange because there is no evidence that narrow QRS type II block is less serious than wide QRS type II block. The statement that "type II block is usually infranodal especially when the QRS is wide" may be the basis for this potentially misleading distinction. Type II block according to the strict definition is always infranodal and should be a class I indication regardless of QRS duration, symptoms or whether it is paroxysmal or chronic.

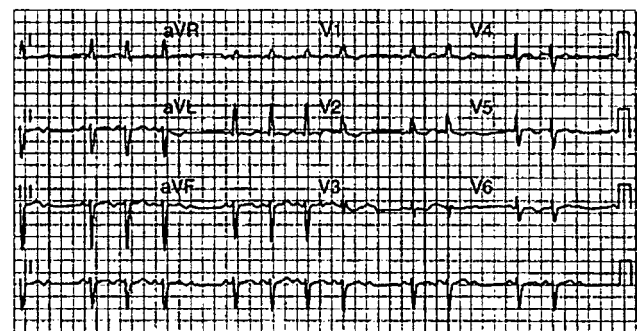


FIGURE 6. Sinus rhythm with second-degree Type II AV block in the presence of right bundle branch block and left anterior hemiblock. There are tiny q waves in V_2 and V_3 probably related to left anterior hemiblock rather than old anterior myocardial infarction. Note that the sinus rate is constant and the PR interval after the blocked beat remains unchanged. (Barold SS. Pacemaker treatment of bradycardias and selection of optimal pacing modes. In: Zipes DP (Ed.). Contemporary Treatments in Cardiovascular Disease, 1997; 1:123, with permission).

The literature on the diagnosis of type II block is replete with errors because the diagnostic importance of the rate criterion and need for an unchanged PR interval after a single blocked impulse are often ignored [3] (Fig. 7). A constant PR after the blocked beat is a *sine qua non* of Type II block. The diagnosis of Type II cannot be made if the P wave after a blocked impulse is not conducted with the same PR interval as all the other conducted P waves. The shorter PR interval after a single blocked P wave may either be due to improved conduction (Type I block) or AV dissociation due to an escape AV junctional beat that bears no relationship to the preceding P wave. In other words, Type II second-degree AV block cannot be diagnosed whenever shortened AV interval occurs after the blocked P wave. In such a situation the pattern is either Type I or unclassifiable. Type II block is sometimes described as having all the conducted PR intervals are constant. There is an important loophole in this statement. It could be interpreted that the behavior of the first P wave after the blocked impulse can be disregarded in the diagnosis (Fig. 3A, 8). If the P wave is absent there is no opportunity to determine whether the behavior of the first PR interval after the blocked impulse and the diagnosis of type II block cannot be established.

Site of Block

Type II according to the strict definition occurs in the His-Purkinje system and rarely above the site of recording of the His bundle potential in the proximal His bundle or nodo-

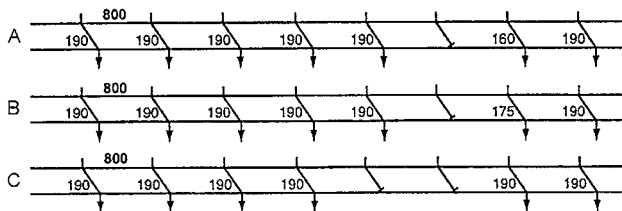


FIGURE 7. Diagrammatic representation of various forms of second-degree AV block with the same format as in Fig 2. (A) Dropped beat followed by a 30ms shortening of the PR interval. This pattern should not be called type II AV block. It may be a type I AV block or unclassifiable if shortening of the PR interval is due to an AV junctional escape beat. (B) Type II AV block according to some of the old definitions. This is now labeled as a type I with very small increments in conduction. Some workers still call this arrangement type II AV block. The diagnosis of type II block cannot be made if the PR interval after the blocked beat is not equal to all the other PR intervals. (C) Advanced second-degree AV block (failure of conduction of 2 consecutive P waves without warning). All the PR intervals are constant including the first one after the block. This suggests infranodal AV block. Some workers cling to the original Mobitz definition and call this sequence type II block. Reproduced with permission from reference 11.

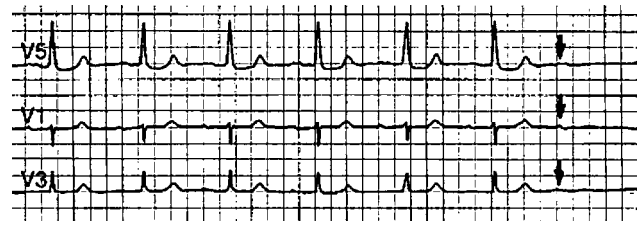


FIGURE 8. Narrow QRS type I block registered in a 3-lead Holter recording. There is sinus arrhythmia. The last 3 PR intervals before the blocked beat (arrow) are constant. This pattern should not be classified as type II block when conduction of the post-block P wave is not seen. Actually the P wave after the block was conducted with a shorter PR interval consistent with type I block.

Hisian junction. Type II block has not yet been convincingly demonstrated in the N zone of the AV node [3]. Most if not all the purported exceptions involve reports where type I blocks (shorter PR interval after the blocked beat) are claimed to be type II blocks by using loopholes in the definitions of second-degree AV block. Because type II invariably occurs in the His-Purkinje system, it should be a class I indication for pacing.

TYPE II SECOND-DEGREE AV BLOCK: TRUE OR FALSE?

When confronted with a pattern that appears to be type II with a narrow QRS complex (especially in Holter recordings), one must consider the possibility of type I block without discernible or measurable increments in the PR intervals. Sinus slowing with AV block rules out type II block. Vagal AV block (discussed later) rarely involves more than block of 2 consecutive P waves. Difficulty arises when the sinus rate is stable. When a type II-like pattern with a narrow QRS complex occurs in association with type I sequences, true type II block can be safely excluded because the co-existence of both types of block in the His bundle is almost unknown (Fig. 4). True narrow QRS type II block occurs without sinus slowing and is typically associated with sustained advanced second-degree AV block far more commonly than type I block in association with true type II block. In other words, AV conduction ratios $>2:1$ (3:1, 4:1) AV block are rare in vagal block [12].

FIXED-RATIO AV BLOCK

2:1 AV block. 2:1 AV block can be AV nodal or in the His-Purkinje system. It cannot be classified as type I or type II block because there is only one PR interval to examine before the blocked P wave (Fig. 9). 2:1 AV block is best labeled simply as 2:1 block [3,15]. For the purpose of classification according to the World Health Organization and the ACC, it is considered as “advanced block” as are 3:1, 4:1 etc. AV block. Confusion arises when the term “advanced AV block” (defined in the ACC/AHA guidelines as a form of second-

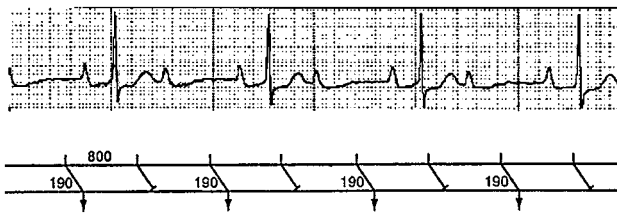


FIGURE 9. Fixed 2:1 AV block. This cannot be classified as type I or type II block. Reproduced with permission from reference 11.

degree AV block of 2 or more P waves) is used to describe both second- and third-degree AV block [1].

The site of the lesion in 2:1 AV block can often be determined by seeking the company 2:1 AV block keeps. An association with either type I or type II second-degree AV block helps localization of the lesion according to the correlations already discussed. Outside of acute myocardial infarction, sustained 2:1 and 3:1 AV block with a wide QRS complex occurs in the His-Purkinje system in 80% of cases and 20% in the AV node [3]. It is inappropriate to label AV nodal as 2:1 or 3:1 AV block as type I block and infranodal 2:1 or 3:1 AV block as type II block because the diagnosis of type I and type II blocks is based on electrocardiographic patterns and not on the anatomical site of block.

When stable sinus rhythm and 1:1 AV conduction is followed by sudden AV block of several impulses (>1), and all the PR intervals before and after the block remain constant strongly suggest infranodal block and the need for a pacemaker. This arrangement is sometimes called Type II block although it does not conform to the accepted contemporary definition of type II block. The purist will insist on calling this pattern (3:1, 4:1 AV block) type II AV block by citing the original description by Mobitz despite the accepted contemporary definitions that such patterns should not be labeled type II AV block [16] (Fig. 10). When the first PR interval after the blocked P waves (in 3:1, 4:1 AV block) is not equal to previous PR intervals the block can be either in the AV node or the His-Purkinje system.

Paroxysmal AV block has been defined as the abrupt occurrence or repetitive block of the atrial impulses with a relatively long (approximately 2 seconds or more) ventricular asystole before the return of conduction or escape of a subsidiary ventricular pacemaker [17]. We believe that this form of AV block does not represent a separate entity and is best

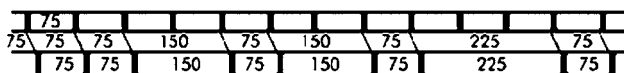


FIGURE 10. Diagrammatic representation of second-degree type II AV block from Mobitz's original article [16].

considered simply as advanced or complete block.

FIRST-DEGREE AV BLOCK

It is now recognized that even an isolated markedly long PR interval can cause symptoms similar to the pacemaker syndrome especially in the presence of normal left ventricular function [18]. During markedly prolonged anterograde AV conduction, the close proximity of atrial systole to the preceding ventricular systole produces the same hemodynamic consequences as continual retrograde ventriculoatrial conduction during VVI pacing (Fig. 11, 12). This is why symptomatic marked first-degree AV block has been called "pacemaker syndrome without a pacemaker" but we believe that the term "pacemaker-like syndrome" is more appropriate. An AV junctional rhythm with retrograde ventriculoatrial conduction may also produce the same pathophysiology. The 2002 ACC/AHA/NASPE guidelines for pacemaker implantation now advocate pacing in acquired marked 1st degree AV block (>0.30 sec.) as a Class IIa indication [1]. Patients with a long PR interval may or may not be symptomatic at rest. They are more likely to become symptomatic with mild or moderate exercise when the PR interval does not shorten appropriately and atrial systole shifts progressively closer towards ventricular systole. The class II recommendation does not really apply to patients with congestive heart failure, dilated cardiomyopathy, and marked first-degree AV block where biventricular pacing would be more beneficial than conventional dual chamber pacing. The clinician must decide in the individual patient whether there is a net benefit provided by 2 opposing factors: a positive effect from AV delay optimization and a negative

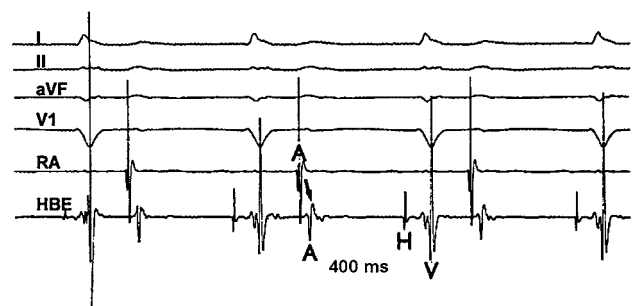


FIGURE 11. Surface ECG and intracardiac recording from a patient with symptomatic marked first-degree AV block originally misdiagnosed as having as AV junctional rhythm with retrograde VA conduction. RA = high right atrial electrogram, HBE = Electrogram at site of His bundle recording. Note the sequence of atrial activation (RA to HBE) is consistent with sinus rhythm and rules out retrograde atrial activation. The AH interval reflecting AV nodal conduction) is markedly prolonged. The patient had a normal left ventricular ejection fraction and complained of exertional dyspnea.

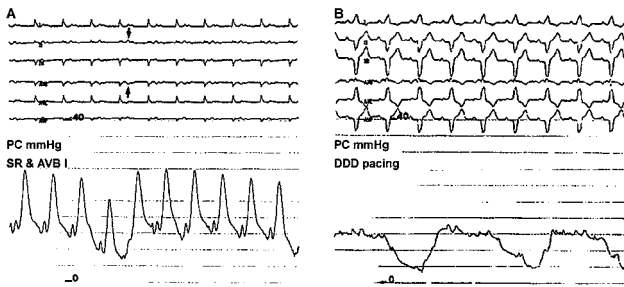


FIGURE 12. A: Same patient as Figure 10. Pulmonary capillary wedge pressure shows large cannon waves. Scale 0-40 mm Hg. B: Same patient after testing with a temporary dual chamber pacemaker with a physiologic AV delay. Note the normal pulmonary capillary wedge pressure. The patient was markedly improved after the implantation of a dual chamber pacemaker with AV delay optimization.

effect impact of reduced left ventricular function from aberrant pacemaker-controlled depolarization (Fig. 12). A recent study suggests that improvement with dual chamber pacing becomes evident with a PR interval > 0.28 sec [19].

INTRAVENTRICULAR CONDUCTION AND PROVOCABLE AV BLOCK BLOCKS

Although the meaning of bifascicular block is obvious, that of trifascicular block is not as simple. The term trifascicular block is often used rather loosely as previously emphasized. Bilateral bundle branch block, despite 1:1 AV conduction, carry a poor prognosis and should be a Class I indication for pacing, even in an asymptomatic patient.

1. EXERCISE

Permanent pacing is recommended as a class I indication in symptomatic or asymptomatic patients with exercise-induced AV block (absent at rest) because the vast majority are due to tachycardia-dependent block in the His-Purkinje system and carry a poor prognosis [20-22] (Fig. 13). This form of AV block is often reproducible in the electrophysiology laboratory by rapid atrial pacing because it is tachycardia-dependent and rarely due to AV nodal disease. Exercise-induced AV block secondary to myocardial ischemia is rare and does not require pacing unless ischemia cannot be alleviated [23].

2. DURING AN ELECTROPHYSIOLOGIC STUDY

When an electrophysiologic study is performed for the evaluation of syncope, many workers believe that AV block or delay in the following circumstances constitutes an indication for permanent pacing. a) A markedly prolonged HV (from His bundle potential to earliest ventricular activation) interval (≥ 100 ms; normal = 35-55 ms) identifies patients with a higher

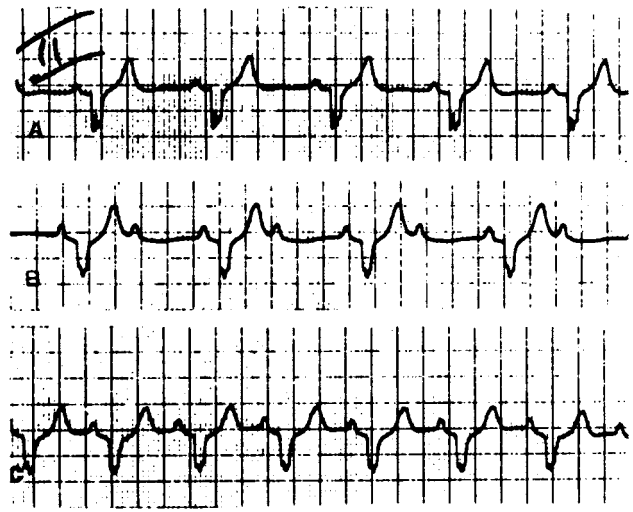


FIGURE 13. A. Lead II ECG in a patient with syncope intra-ventricular conduction block but no documented AV block at rest. B. On exercise 2:1 AV block occurs and 1:1 AV conduction returns during the recovery period. A subsequent electrophysiologic study revealed infranodal AV block at an atrial pacing rate of 110/min and a permanent pacemaker was implanted. No further syncope occurred.

risk of developing complete AV block and need for a pacemaker [1] (Fig. 14). A study can define by a process of exclusion which patients might benefit from pacing in the presence of HV prolongation (≥ 70 ms) and no other electrophysiologic abnormality such as inducible ventricular tachycardia. b) The development of second- or third-degree His-Purkinje block in an electrophysiologic "stress test" performed by gradually increasing the atrial rate by pacing is an insensitive sign of conduction system disease but constitutes a class I indication for pacing because it correlates with a high incidence of third-degree AV block or sudden death [24]. c) Bradycardia-dependent (phase 4) block (not bradycardia-associated as in vagally-induced AV block) is rare and always infranodal. It can be evaluated with His bundle recordings by producing bradycardia and pauses by the electrical induction of atrial or ventricular premature beats. d) A drug challenge with procainamide that depresses His-Purkinje conduction may be used to provoke HV interval prolongation or actual His-Purkinje block (according to published criteria) in susceptible patients and define the need for a pacemaker [25].

PERMANENT PACING FOR AV BLOCK AFTER ACUTE MYOCARDIAL INFARCTION

The requirement for temporary pacing in acute myocardial infarction (MI) does not by itself constitute an indication for permanent pacing. Unlike many other indications, the need for permanent pacing after acute MI does not necessarily depend on the presence of symptoms

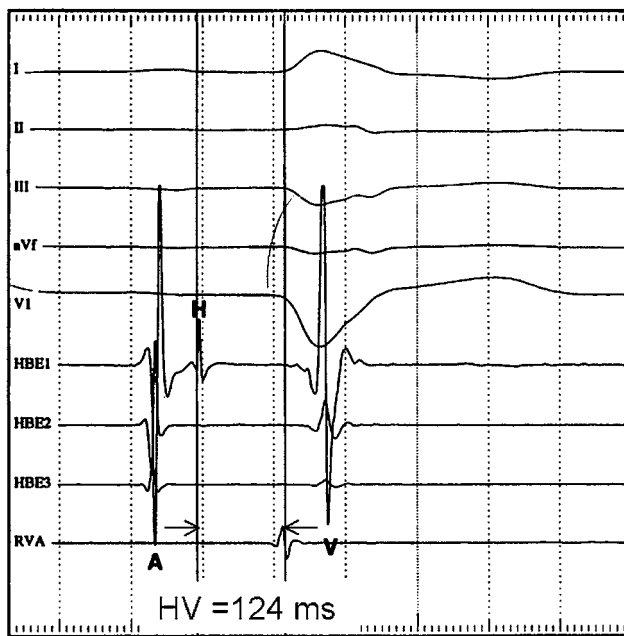


FIGURE 14. Very long HV interval representing severe disease of the His-Purkinje system. His bundle recording in a patient with right bundle branch block and syncope. Left ventricular ejection fraction was normal. There was no documentation of second- or third-degree AV block before the electrophysiologic study. Note the very long HV interval of ms measured from the His bundle potential to the earliest ventricular activation either in the surface or intracardiac leads. (normal = 35-55 ms) responsible for the first-degree AV block. Time lines = 10 ms. A = low atrial depolarization, H = His bundle potential, V = Activation of high ventricular septum. Syncope disappeared after implantation of a permanent pacemaker.

Acute Inferior Myocardial Infarction

Permanent pacing is almost never needed in inferior MI and narrow QRS AV block. Pacemaker implantation should be considered only if second- or third-degree AV block persist for 14-16 days [26,27]. The use of permanent pacing is required in only 1-2 % of all the patients who develop acute second or third-degree AV block regardless of thrombolytic therapy. Narrow QRS type II second-degree AV block has not yet been reported in acute inferior MI [28-31].

Acute Anterior Myocardial Infarction

Patients who develop bundle branch block and transient second- and third-degree AV block during anterior MI have a high in-hospital mortality rate and are at a high risk of sudden death after hospital discharge. Sudden death usually is due to malignant ventricular tachyarrhythmias and less commonly related to the development of complete AV block with prolonged ventricular asystole. The use of permanent pacing in patients with transient trifascicular AV block or bilateral

(alternating) bundle branch block is still controversial, but most workers recommend it with the aim of preventing sudden death from asystole despite the return of 1:1 AV conduction. Permanent pacing is not indicated in patients with acute anterior MI and residual bundle branch or bifascicular block without documented transient second- or third-degree AV block because there is no appreciable risk of late development of complete AV block. Measurement of the HV interval does not predict which patients will develop progressive conduction system disease.

Patients with an anterior MI who require permanent pacing often have a low left ventricular ejection fraction that makes them potential candidates for a prophylactic implantable cardioverter-defibrillator. A recent study in patients who suffered an acute MI suggests waiting 3 to 6 months before implanting a defibrillator [32]. Despite this recommendation, it makes sense to implant a cardioverter-defibrillator (which contains a pacemaker component) in patients who actually require only a permanent pacemaker at that juncture. Such patients are also at risk for sudden death from a ventricular tachyarrhythmia. It makes no sense to wait 3 to 6 months without protection for bradycardia until a cardioverter-defibrillator can be implanted on the basis of a poor LV ejection fraction according to the DINAMIT [32].

VAGALLY MEDIATED AV BLOCK

Vagally mediated AV block is generally a benign condition that can superficially resemble type II block. This phenomenon has been called "apparent type II block" because it simulates type II block but it is generally considered a type I variant [3,33]. Vagally mediated AV block occurs in the AV node and differs from neurally mediated (malignant vasovagal) syncope where head-up tilt testing causes sinus arrest and rarely predominant AV block. Vagally induced AV block can occur in otherwise normal individuals and also in patients with cough, swallowing, hiccups, micturition, etc. when vagal discharge is enhanced (Fig. 4). Electrophysiologic studies in vagally-mediated AV block are basically normal. Vagally mediated AV block is characteristically paroxysmal and often associated with sinus slowing. As a rule, AV nodal block is associated with obvious irregular and longer PP intervals and is bradycardia associated (not bradycardia dependent), i.e., both AV block and sinus slowing result from vagal effects. An acute increase in vagal tone may occasionally produce AV block without preceding prolongation of the AH interval (constant PR), giving the superficial appearance of a Type II AV block mechanism, i.e., no PR prolongation before the blocked beat. In this situation, AH prolongation may occur during the initial several beats when AV conduction resumes. Vagally induced block is occasionally expressed in terms of constant PR intervals before and after the blocked impulse, an arrangement that may lead to an erroneous diagnosis of the more serious type II block if sinus slowing is ignored (Fig.

4). Sinus slowing can sometimes be subtle because the P-P interval may increase by as little as 0.04 sec.

AV BLOCK IN ATHLETES

Severe sinus bradycardia and third-degree AV block can occur at rest or after exercise in athletes and lead to symptoms such as lightheadedness, syncope, or even Stokes-Adams attacks. These changes are considered secondary to increased parasympathetic (hypervagotonia) and decreased sympathetic tone on the sinus and AV node related to physical training [34]. Most patients become asymptomatic after physical deconditioning. If the latter produces no response or the patient refuses to decrease athletic activities, a permanent pacemaker becomes indicated. Some of the so-called "athletic patients" improved by pacing represent individuals who would otherwise benefit from pacing, i.e., subjects with sinus node disease rendered symptomatic by increased vagal tone related to training or athletes with spontaneous or exercise-induced infranodal block.

Atrioventricular block in athletes is most probably an expression of hypervagotonia. This form of AV block may or may not be associated with sinus bradycardia because the relative effects of sympathetic and parasympathetic systems on the AV and sinus node may differ. AV block in athletes responds to exercise or atropine. A number of authors have indicated Mobitz Type II second-degree AV block (sometimes called Mobitz AV block as opposed to Wenckebach AV block) can occur in young athletes. The diagnosis of Type II AV block immediately raises the question of a permanent pacemaker, especially in symptomatic patients. We believe that Mobitz Type II second-degree AV block (always infranodal) does not occur in otherwise healthy athletes. The purported occurrence of Type II AV block in some reports appears related to failure of applying the correct definition of Type II second-degree AV block.

CONCLUSION

Second-degree AV block remains poorly understood despite the major advances in cardiac electrophysiology in the last 35 years. The literature abounds with varying definitions of second-degree AV block especially Mobitz Type II block. It should therefore not be surprising that during formal testing, physicians score more poorly with second-degree AV block ECGs than with those of other arrhythmias [35]. Indeed it was stated a few years ago that "Mobitz II block is misunderstood more than any other abnormality of rhythm or conduction" [36]. Much of the prevailing confusion surrounding second-degree AV block would disappear with the proper application of strict and uniform definitions. The understanding of second-degree AV block is basically an exercise in clinical logic that centers on appreciating the definitions of type I and type II block.

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